effective whereas the latter was not. Clinical trials of Brazilian, Ecuadorian and possibly Colombian antivenoms are planned in the Amazon region of Ecuador in the near future.

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Comparison of $F(ab')_2$ and Fab efficiency on plasma extravasation induced by Viper aspis *venom.* M. Sorkine,[1,2] B. Saliou[1] and C. Bon[1] ([1]Unité des Venins, Institut Pasteur 25, Rue du Dr Roux, 75724 Paris Cedex 15, France; and [2]S.A.R., Hôpital Henri Mondor, Créteil 94000, France).

Envenomation caused by European vipers associates local signs, essentially oedema and systemic manifestations. Extensive oedema produces pain and inability to use the affected limb, and is a major factor of hypovolemia. Since symptomatic treatment failed to prevent this oedema, the effect of antivenom on plasma extravasation, the first step of oedema formation, was examined. The purpose of the study was to compare in a mouse model the effect of $F(ab')_2$ (equine) and Fab (equine and ovine) on capillary permeability increase (CPI) induced by *Vipera aspis aspis* venom. $F(ab')_2$ (ID_{50} 2 mg/kg) and Fab (ID_{50} 2.5 mg/kg) reduced considerably CPI when mixed with venom prior to intradermal injection. When fragments were intravenously injected before intradermal administration of the venom, a larger amount of fragments was necessary, Fab being five times more effective than $F(ab')_2$ (ID_{50} 105 mg/kg compared to ID_{50} 520 mg/kg). Furthermore, immunoglobulins injected after the venom $F(ab')_2$ were ineffective, while Fab has a residual effect (ID_{50} 235 mg/kg). No difference was observed on the efficiency of ovine and equine Fab. These data showed firstly that the *in vitro* neutralization of the venom by immunoglobulin fragments does not reflect their *in vivo* efficiency. Secondly, Fab was considerably more effective than $F(ab')_2$ in reducing CPI induced by venom. One explanation is the different kinetics of these fragments. The smaller size of Fab results in faster diffusion and a greater volume of distribution.

Molecular structure and action mechanism of the specific crotoxin inhibitor from Crotalus durissus terrificus *serum.* J. Perales,[1] C. Villela,[1] G. Domont,[1] V. Choumet,[2] B. Saliou,[2] H. Moussatché[1], C. Bon[2] and G. Faure[2] ([1]Departamento de Fisiologia e Farmacodinâmica, Instituto Oswaldo Cruz, Rio de Janeiro, Brazil; and [2]Unité des Venins, Institut Pasteur, Paris, France).

An antivenom protein component that specifically neutralizes crotoxin, the main lethal component of rattlesnake